Environmental Health Perspectives
Vol. 63, p. 249, 1985

Letters to the Editor

Dear Sirs:

The authors reviewed the growth-promoting and carcinogenic effects of raw soya flour in rats and found that if raw soya flour-containing diets are fed for more than one year, almost 10% of animals develop pancreatic cancer. In addition, by eating raw soya flour they markedly potentiate the action of subthreshold amounts of pancreatic carcinogens. Therefore, they consider the raw soya flour a potent promoter as well as a weak carcinogen. To explain these phenomena, the authors focus their attention on the trypsin inhibitor contained in raw soya bean food and they strongly urge the appropriate national regulatory authorities to test the carcinogenic and promoter effects of soybean food products.

I wish to mention that soybeans are a very important food in Oriental countries, especially in China (soybean is also called Chinese bean). If we admit that the soybean food diet is an important factor in inducing pancreatic carcinoma, how can we explain the lower incidence of pancreatic cancer in China as compared to America (Robert W. Miller, Epidemiology. In: Cancer in China [Henry S. Kaplan and Patricia Jones Tsuchitani, Eds.], Alan R. Liss Inc., New York, 1978, pp. 39–57)? In fact, trypsin inhibitors are also present in cow's milk. Fortunately, both in soya bean and in milk most of these inhibitors are destroyed by various means of heating and cooking.

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Dear Sirs:

We read with great interest an article in Environmental Health Perspectives (60: 405–410, 1985) titled "Measurement of Nicotine in Building Air as an Indicator of Tobacco Smoke Levels" by Don C. Williams, John R. Whitaker, and Walter G. Jennings. Unfortunately, there are conclusions drawn from the measurements that appear to be in gross error. First, the authors do not explain how they arrive at a passive cigarette exposure of 1.1 cigarettes. However, assuming that the authors' calculations from the pump collection are correct and the concentration of nicotine in the "smoky office" is 1.96 $\mu g/m^3$, then the calculated nicotine exposure based upon a mainstream value of 1.0 mg nicotine/cigarette and a respiration volume of 1.0 m^3/hr (Repace and Lowrey, Environment International, 11: 14, 1985) is 0.0156 cigarette equivalents per 8-hr day. This exposure is much less than the 1.1 value cited in the paper.

Second, the concentration of nicotine quoted in the pump collection experiment also appears to be in error. The authors' value of 1.96 $\mu g/m^3$ seems to have been calculated by dividing the nicotine collection rate by the rate the air was pumped over the plate. This is incorrect. With the value of nicotine collection rate determined by the authors, 180 ± 7 $pg/m^2/min$, the surface area of the Petri dish, $6.22 \times 10^{-3} m^2$, and the volume of air pumped over the Petri dish, $1.72 \times 10^{-2} m^3$, it can be shown that the highest concentration of nicotine determined in the room was 1.22×10^{-2} $\mu g/m^3$. This converts to an 8-hr exposure of 0.0000976 cigarette equivalents! According to the results of Muramatsu and coworkers (Environmental Research 35: 218–227, 1984), the levels of nicotine in typical office areas range from 10 to 30 $\mu g/m^3$. The highest levels of nicotine cited in the current paper are significantly lower than those of Muramatsu. This suggests that either the office case cited in this paper has exceedingly clean air or the reported novel collection method *is very inefficient.*

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